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ABSTRACT

This paper examines the effects of unavoidable pollutants on fetal development in humans. Inevitable pollutants such as radiation, pesticides, gases and lead found in the air, water, and food of our industrialized society are discussed as well as psychological correlates of industrialization and urbanization such as stress, increased noise levels and crowding. The paper puts research published primarily within the fields of medicine and epidemiology in a developmental context, suggesting how even subtle fetal impairment can affect cognitive, social, and perceptual development both before and after birth. (JMB)

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EFFECTS OF INEVITABLE ENVIRONMENTAL POLLUTANTS

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Effects of Inevitable Environmental Pollutants

Recent research has shown that maternal malnutrition, alcoholism and drug addiction have adverse effects on fetal development (Lester, 1977; Winsmore, 1977). This paper will focus on substances which can also affect fetal development, substances which individual pregnant women cannot easily avoid. The paper will first identify these inevitable pollutants and then will discuss the interaction between even subtle fetal deviations and the process of development. It will include substances found in the air, water and food of our industrialized society, and psychological correlates of industrialization and urbanization: stress, increased noise levels and crowding. These factors as well as chemical pollutants can affect the fetus. For example, population density has been found to relate significantly to serious cardiac defects at birth (Rothman & Fyler, 1977).

This paper will put research published primarily within the fields of medicine and epidemiology in a developmental context, and suggest how even subtle fetal impairment can affect cognitive, social, and perceptual development both before and after birth.

Nine out ten women will work outside of their homes at some time in their lives, and the majority of these will be in their child bearing years (U.S. Department of Labor, Women's Bureau, 1974). Thus, we must pay special attention to the dangers to which pregnant working women are exposed. A majority of women work for economic reasons and cannot stop work while they are pregnant, certainly not during the crucial first trimester. It is potentially easy to document the nature and concentration of a pollutant to which workers are exposed and then to trace its effects on reproductive patterns. In fact, in the cases of substances like x-ray, Kepone, gases, and lead exposure, careful monitoring has revealed that both women workers and the wives of male workers have had increased incidents of sterility, spontaneous abortion, and birth defects (Hricko & Brunt, 1976). Such controlled and work-related research has only begun and more attention must be paid to health and safety standards on the job especially for women workers.

Studies in the workplace are somewhat easier to document, conduct and control, than those outside the workplace. However, it is not only workers who are exposed to the polluting substances. Many of these

pollutants are air or water borne and are emitted as industrial by-products exposing the neighboring communities. One study found elevated lead content in children who lived in housing which was free from lead paint, but near a major lead processor (Environmental Health Perspectives, 1974).

Effects on the Fetus

Mutagens

Fetal development can be affected several ways. Some substances act as mutagens. Others cause structural malformations. And some retard growth, resulting in low birth weight infants (Wilson, 1973). Mutagens cause a change in the genetic material of living cells. Mutagens can affect either the male sperm cell or a female egg cell. The defect may be passed on to all of the infant's children, and chromosome aberrations are found in 30% of spontaneous miscarriages (Sutton, 1974).

One of the most frightening aspects of mutagens is that the exposure to the mutagenic substance can occur long before the fetus is conceived. This can be seen dramatically in the cases of radiation, pesticides, various gases, and lead.

Radiation is one of the best known mutagens. Among pregnant women exposed to the atomic bomb explosions at Nagasaki and Hiroshima there was a high incidence of perinatal death and mental retardation in surviving children (Wilson, 1973; Ferreria, 1969; Blot, 1975). Infants who have been irradiated in utero as a result of maternal x-rays are found to have severe nervous system defects, are mentally retarded, and at risk for childhood leukemia(Wilson, 1973; Ferreria, 1969).

There is a growing concern over the potential mutagenic effects of low level radiation present in our environment. A study completed in 1955 (before widespread testing of nuclear weapons and nuclear power plants) found a significant relationship between the radioactivity level of a township's water supply and the incidence of infants malformed at birth (Gentry, Parkhurst, Bulin, 1955). Several studies have linked paternal exposure to radioactivity or radar with congenital abnormalities in children (Kitabatake, Watanabe & Sato, 1974). And recent news reports suggest that radioactive fallout from China may be responsible for the death of 300 American infants.

Radiation is not the only mutagen. Kepone, a pesticide, has been found to cause sterility in men. More than half

of the employees of a Kepone manufacturing plant in Virginia developed symptoms of Kepone poisoning, including infertility and loss of libido (Hricko & Brunt, 1976). Studies concerned with the more general relationship between pesticides and birth defects appear to find a relationship but are not totally conclusive (Wilson, 1973). There have been no studies of the effect of low level, long-term exposure on human fetuses. However, DDT, another pesticide, has been detected in the blood of newborns in the Mississippi Delta, even though none of the mothers had been employed as farm laborers during the previous two years (D'Ercole, Arthur, Cain & Barrentine, 1976).

Both male and female operating room workers who have constant exposure to anesthetic gases have been found to be at risk for mutagenic effects. Wives of male operating room workers have one and a quarter greater risk of having a child with birth defects than wives of unexposed non-operating room personnel. And wives of dentists exposed to anesthetic gas have one and three quarters greater risk than wives of unexposed dentists. The risk of birth defects for female operating room workers is even higher: up to two times greater than non-exposed medical personnel and the risk for miscarriages is up to three times greater for exposed females (Cohen, Belville & Brown, 1971; American Society of Anesthesiologists, 1974).

Another gas which disrupts fetal development is vinyl chloride which is released during the production of various plastic products and during their decomposition by fire. Several studies find a significantly higher incidence of fetal death among the wives of workers exposed to vinyl chloride than wives of controls (Infante, McMichael, Wagoner, Waxweiler & Falk, 1976; Selikoff, 1974).

Males, exposed to chronic levels of lead, according to an early study, have twice as many childless marriages as would be expected (Hamilton, 1934). In a more recent study 90% of the workers exposed to high levels of lead had sperm abnormalaties while workers exposed to lower levels showed decreased fertility, or decreased ability to produce healthy sperm (Hricko & Brunt, 1976). Thus, radiation, chemicals, and pesticides are mutagens which can affect conception and physical fetal development.

Other Teratogens

Other teratogens interfere with the development of the fetus following conception. The resulting abnormal development can cause miscarriage or congenital defects.

These defects may be present at birth or only later become evident. The identification and classification of these teratogens is a complex matter. Many substances have no observable effect on the pregnant woman. She may be unaware of being exposed; yet the immature fetus, lacking the capacity to metabolize and detoxify noxious substances, is damaged. For example, a pregnant woman who is immune to rubella may become the vehicle for fetal infection and resulting abnormality including heart defects and deafness (Ferriera, 1969). Other unobservable substances can affect fetal growth. For example, while high levels of carbon monoxide result in stillbirths or severe retardation, long-term, low-level exposure is linked to low birth weight infants (Hricko & Brunt, 1976). Carbon monoxide is a by-product of smoking, and non-smoking women are exposed to it in poorly ventilated rooms, in traffic jams through exhaust fumes, and in areas of industrial smog. A recent and important study illustrates the effects of carbon monoxide and other air pollutants on fetal growth. Williams, Spence and Tideman (1977) found that the air pollution levels in different parts of the Los Angeles Basin accounted for significant variance in baby birth weights. In fact, mothers who lived in the most polluted areas of the city gave birth to babies whose average weight was eleven ounces lower than

that of infants whose mothers lived in less polluted areas. Pregnant women are also exposed to low levels of lead through automobile exhausts, water supply and industrial fumes. Lead can act as a teratogen in the forms of low birth weight and later learning problems (Sontag, 1975; Angle & McIntire, 1969).

Interactions Between Substances

Teratogens can interact with each other to produce fetal damage. Substances which are not damaging to fetal development in themselves or are not dangerous at low dosages may become so when combined with other substances. Prenatal exposure to chronic low dosage is unlikely to result in clinically evident birth defects (Spykes, 1975). However, the effects of such exposure can have subtle long-term effects. Low level exposure to one substance in combination with exposure to another could be very damaging.

Research in the area of teratogen interaction is in its prelimiary stages, but the data which do exist are provocative. Such research is especially difficult to conduct because we cannot generalize directly from animals to humans. For example, thalidomide does not

cause malformations in fetal rats while it is an extreme teratogen in humans and some other primates. We shall report results of animal research as suggestive for humans. In one study (Yeilding, Riley, Yeilding, 1976), caffeine and chloroquine, an antimalarial drug, were administered to pregnant mice at levels known to be non-teratogenic. The mice were then exposed to x-rays. The chloroquine and caffeine treated mice had double the birth defects (cleft palate and tail abnormalaties) of the mice exposed only to x-rays.

Another set of studies suggests a teratogenic interaction between aspirin and benzoic acid, a food preservative. Large doses of aspirin, much greater than women normally take, are known to produce abortions and malformations in mice and monkeys (Wilson, 1973). Two retrospective human studies found that significantly more women delivering malformed infants have taken aspirin during the first trimester than women delivering normal babies, although another retrospective study found the reverse relationship (Wilson, 1973). When pregnant rats were given moderate level doses of aspirin plus benzoic acid, the preservative, the number of fetal malformations increased (Wilson, 1973). Similar metabolic interaction between benzoic acid and aspirin are known to occur in humans (Wilson, 1973).

Cumulative Effects

Pollutants may have indirect effects. Industrial wastes, pesticides, food and fuel additives as well as other environmental pollutants to which humans are regularly exposed can accumulate within the body. Prenatal exposure compounded by exposure as an infant and child may mean that the teratogen reaches critical levels only later in the organism's life (Spykes, 1975). Lead and the chemicals found in pesticides are among the substances known to accumulate (Spykes, 1975; D'Ercole et al.,1976). High levels of lead are associated with hyperactivity and brain damage (Hricko & Brunt, 1976). The effects of the accumulation of chlorinated hydrocarbons found in pesticides are not known, but acute toxic effects include tremors, delerium, seizures, coma, and death (D'Ercole et al.,1976).

Exposure to a substance in utero, when the organism is less able to ward off damage, may sensitize the offspring to later exposure. Pregnant mice were exposed to sublethal doses of microwaves and then gave birth normally (Rugh, 1976). When the infant mice were two months old

they were exposed, continuously until death, to the same low doses of microwaves. These mice were compared to a group of mice not irradiated *in utero*. The mice exposed *in utero* were killed more quickly by the microwaves.

Further evidence for sensitizing effect comes from the effect of DES on humans. DES is a synthetic estrogen used as a morning-after pill, as therapy to prevent miscarriages, and as a fattener for cattle and chickens. The daughters of pregnant women who took DES in the fifties to prevent miscarriages have high incidences of vaginal and cervical cancer (Mattingly & Stafl, 1976). There is some thought that this is linked not only to their exposure *in utero* but to exposure as children and adults to estrogen fed beef and chickens. A recent study (Reinish, 1977) also suggests that synthetic hormones may affect personality development. Children exposed *in utero* were found to differ significantly from their own unexposed siblings on the Cattell Personality Questionnaire even when pre- and perinatal complications were controlled. They did not differ on IQ tests.

The effects of many of the substances we have called inevitable pollutants vary with the dosage level to which a pregnant woman is exposed. When the fetus is

exposed to high levels of the substance due to an industrial or environmental accident, the result is usually spontaneous abortion, still-birth, or a congenital malformation. Exposure to low level doses tends to result in low birth weight, small for date infants, or in damage which only becomes apparent with time. Lead was one of the first substances identified as a teratogen. Based on a 1900 study which found that women working with lead had three times elevated miscarriages rate, women were kept out of the lead industry until 10 years ago. Women have traditionally used lead to get rid of unwanted pregnancies (Hricko & Brunt, 1976). More recent studies have shown that exposure to low levels of lead results in low birth weight and impaired learning capacity (Hricko & Brunt, 1976).

Cigarette smoke is another substance which has been suspected of having damaging effects for a relatively long period. More recently, research has established without question that babies born to heavy smokers differ in three important ways from those born to non-smokers. They have lower birth weights. They are more apt to be premature. They have increased risk of perinatal mortality. More specifically, the evidence indicates that babies born to smokers weigh six to eight ounces less

than those born to non-smokers. Moreover, the risk of having a baby who weighs under 2500 grams (5½ pounds) doubles for smokers. This means that approximately 30% of all such low birth weights can be attributed to maternal smoking. These data hold even when maternal parity, weight, height, age and SES and neonatal sex are controlled statistically. Similar effects of smoking exist for length of gestation. In this instance, the risk of delivering before the thirty-eighth week is one-third higher among smokers than non-smokers. Finally, the risk of perinatal loss increases by approximately 20% among smokers (Meyer, Jonas & Tonascia, 1976). Current research suggests that these effects may be due not only to increased carbon monoxide levels among smokers but also to a decrease in serum vitamin B-12 available to the fetus (Pettigrew, Logan & Willocks, 1977). Another explanation suggests that smokers eat less than non-smokers and hence gain less weight during pregnancy (Davies, Gray, Ellwood & Abernathy, 1976). Whatever the mechanism, however, maternal smoking is a pollutant with strong effects on fetal growth.

The teratogenic effect of low doses of some pollutants may cause alterations in neurodevelopment which will not be evident except as subtle behavioral disturbances later

in life. There is increasing evidence that the brain is more sensitive to foreign chemicals than previously suspected and toxic effects may show up much later in life (Spykes, 1975). This point was demonstrated in a longitudinal study of mice exposed as fetuses to low levels of mercury (Spykes, 1975), which at high levels is associated with miscarriage, mental retardation, seizures, and cerebral palsey in humans (Koos & Longo, 1976). At weaning the majority of the mice appeared normal. By 30 days of age the exposed mice showed less exploratory behavior than controls and were deviant in walking and swimming tests. As adults they showed neurological impairment, and high rates of infections and premature deaths.

Psychological Aspects

Our review thus far has focused on physical effects from pollution. As psychologists, we shall also discuss the ways in which fetal development can be affected by the mother's emotional state, and then put this research in the context of fetal sensory development and infant psychosocial development.

Research on the relation between maternal emotions and fetal outcome suggests that extreme anxiety or stress

during pregnancy is related both to abnormalities at birth and to later behavior. One study (Gorsuch & Key, 1974) indicates that, during the first trimester, anxiety, as measured by an inventory, is related to abnormalities such as prematurity and low birth weight. During the second and third trimesters, however, stressful life events (like death of the husband) rather than anxiety predict abnormalities. Maternal emotions are also related to postnatal behavior. Infants born to mothers who were highly anxious during their pregnancies cried more than those of less anxious mothers during the first days of life (Ottinger & Simmons, 1964). And a retrospective study of 1300 four-year-olds (Stott & Latchford, 1976) found that several types of prenatal stress predicted developmental and behavioral difficulties by age four. For instance, children of women whose pregnancies were characterized by fears about quarrels or money were reported to have twice the number of developmental problems as those of mothers without such fears.

Prenatal Sensory Implications

There has been little research related to prenatal sensory functioning. What evidence does exist suggests that human sensory development in utero is both important

to postnatal functioning and can be influenced by the external environment. During the last trimester, most of the sensory systems are mature and functional (Mistretta & Bradley, 1975). Pregnant women report fetal movements in response to loud noises. In addition, fetal heart rates increase in response to sound stimulations (Sontag, 1970), even when the mother is shielded from the sound (Grimwade, Walker, Bartlett, Gordon, & Wood, 1971). And fetuses have been reported to have been conditioned with auditory stimuli (Spelt, 1948). One indication of the effects of noise pollution on fetal development comes from studies conducted in Japan. Significant variance in birth weight was found to be accounted for by the noise level in areas surrounding the Osaka Airport (Ando & Hattori, 1973). In fact, in the airport area the percentage of birth weights under 3000 grams (6½ pounds) increased significantly between 1963 and 1965 when jet planes began to take off and land. Further research along these lines may reveal other subtle effects of environmental noise on prenatal development. Work with laughing gulls indicates that increased prenatal auditory experience with specific types of parental calls leads to increased activity when the same calls are heard after hatching (Impekoven, 1975). And the Japanese airport research suggests that babies whose mothers lived

near airport noise throughout gestation were less disturbed by that noise during infancy (Ando & Hattori, 1970).

Other prenatal sensory functions are less easy to document experimentally. However, there are some indications of fetal tactile, vestibular, visual, and gustatory functioning (Bradley & Mistretta, 1975; Gottlieb, 1971). Of particular interest would be the effects of increased stimulation in one sensory system (e.g., auditory) on the functioning of not only that system but also the others, both pre- and post-natally. Gottlieb (1971) suggests that there is a developmental interdependence among the senses. Thus, for instance, grasping and thumb sucking in utero might be related to visual-motor development after birth. And, then environmental variation like noise, or taste changes in amniotic fluid could have far-reaching effects.

Neonatal Implications

After birth, the infant's environment becomes far more varied. The normal infant is an active organizer, ready to initiate and to respond to stimuli from the social and nonsocial environment. She can see, hear, taste, and smell. She can visually focus on objects and meet people's

eyes, thereby participating in mutual eye contact. And she can make rudimentary postural adjustments to being held.

The caregiver of the newborn is crucial to the developmental process. A sensitive caregiver optimally regulates environmental stimulation and responds appropriately to the infant's often primitive and subtle initiations. From early in the newborn period, the delivery room and first feeding, there is evidence for the establishment of reciprocal interaction patterns between caregiver and newborn (Brown, Bakeman, Snyder, Federickson, Morgan, & Hepler, 1975). But the infant who has been impaired even in very subtle ways during the fetal period may lack some capacity to act and to respond.

This lack may very well set in motion a spiral of developmental deprivation. Development is an orderly process, building on the interaction between a structured organism and a structured environment. If the organism lacks the ability to act on the environment, this changes the environment and produces a double developmental handicap. For example, in her studies of blind infants, Fraiberg (1974) found that from very early in the newborn period, the caregiver would become upset at the infant's

inability to engage in eye to eye contact. The caregiver would feel as if the child didn't like her; and this often began a cycle of rejection rather than mutual interaction between caregiver and infant.

Drotar and colleagues (1975) also have studied the adaptation of parents to the birth of an infant with a congenital malformation. They found that parents were hesitant to interact with the babies and reluctant to become attached. Interviews with the parents revealed a sequence of parental reactions, including stages of shock, denial and sadness, anger and anxiety. After several months, the parents went through periods of adaptation and attachment resulting in the acceptance and caring for the defective child. This study emphasized the strength of these families in eventually being able to become attached to and accept their child; however, it also points to a disorganized intial response on the part of the caregiver which interfered with establishing early patterns of interaction.

The establishment of these interactive processes with the social and non-social environment is interfered with, for valid medical reasons, by the hospital treatment of infants at risk. The infants are traditionally kept in isolettes, and contact with parents is restricted. There

is some evidence that this restrictive environment produces sensory deprivation. Sensory stimulation programs for low birth weight infants which involve extra handling and visual stimulation have increased later developmental test scores (Scarr-Salapatek & Williams, 1973).

Studies of normal infants and their caregivers suggest that the timing of the first contact between the infant and parent is important (Kennell et. al., 1974, Ringler et. al., 1975, Barnett et. al., 1970, Kennell & Klaus, 1976, Hopkins & Vietze, 1977). The data from these studies suggest that caregiver attachment to the infant is established very soon after birth and that delaying intimate contact between infant and caregiver can lead to caregiver rejection and poor parenting. Studies which link low birth weight infants and later child abuse (Klein & Stern, 1971, Powell, 1970, Schmitt & Kempe, 1975) have suggested that the early separation of caregivers from infants with birth defects may impair parental attachment to them. Several studies have investigated this hypothesis with differing results. Leifer et. al. (1972) found no differences in overt maternal attachment between dyads whose mothers had participated in the caretaking of their premature infants and dyads who had followed the usual pattern of separation. But more parents in the separated group released custody of

the children. Powell (1974) also found no relationship between early contact and ratings of later maternal behavior. A study by Beckwith et. al. (1976) indicates that the situation is more complex than simply the timing and the amount of intial contacts. She was able to differentiate patterns of interaction between mothers and their premature infants. When the infants were assessed at nine months of age those higher in sensory motor skills had had more reciprocal social transactions with their mothers from one month of age and on.

Since the effects of inevitable environmental pollutants are often quite subtle, it is most likely that chronic, low level exposure will produce an infant with subtle deficits. The infants may be less alert and responsive, or exhibit slight behavior abnormalities, or have low, but not alarmingly low birth weights. This is speculation; much more research is needed to support this hypothesis. However, it is clear that even very subtle damage can initiate devlopmental lag. An infant who "isn't quite right" at birth is less able to act on her environment. She thus misses crucial learning experiences and may fail to start in motion reciprocal interaction with the caregiver.

Osofsky (1975) found that more alert and responsive infants have more sensitive and responsive mothers.

Further evidence for our hypothesis comes from a study by Als and colleagues (1976). They investigated the behavior of malnourished infants who had Apgar scores of 7 or above and looked healthy. The researchers concluded that the lack of responsiveness and poor organization of these newborns elicited anxiety in the caregiver which increased the tension of childcare, making interaction difficult.

Conclusion

In conclusion, this paper has suggested that various substances in our environment can affect the developing fetus. Substances like lead, carbon monoxide, noise and industrial chemicals cannot be avoided by pregnant women. Herbicides and pesticides have invaded our food chain. Other pollutants are in our air and water. We, as psychologists have two tasks: First, we must conduct and support research related to environmental effects on fetal development. Second, we must inform our lawmakers of our knowledge and our concern and help them to regulate pollution.

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